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ORIGINAL ARTICLES.

OPHTHALMOPLEGIA EXTERNA COMPLETE WITH PRESERVATION OF ACCOMMODATION AND ACTIVITY OF THE PUPILS.

By DR. S. C. AYRES,

OF CINCINNATI, OHIO.

Mr. E. J. L., aged 38, presents the following appearance: There is ptosis of both eyes. In the right eye the lid droops low enough to partly cover the pupil, but in the left eye it barely escapes the upper edge of the pupil. In looking forward the occipito-frontalis-muscle is brought strongly into action producing marked wrinkling of the forehead. There is absolutely no motion of any of the recti or oblique muscles of either eye. The pupils are active and respond normally to light. There is no weakness of accommodation, as he reads and writes without the aid of glasses. His vision is good in both eyes (0.7 R. and L.), and he has no trouble in seeing in the distance; has never had any asthenopic symptoms. He is a strong, healthy man and comes of healthy stock. He says that his maternal grandfather was similarly affected. His mother told him that the trouble with his eyes came on when he was six years old and was the result of scarlet fever.

There are two doubtful points in the history: first, as to whether the maternal grandfather actually had a similar affection of the ocular muscles; and second, as to whether the pa-

tient's condition was the result of scarlet fever as alleged. If the first statement is true, may it not be congenital? He has brothers and sisters, but none are similarly affected. If he had scarlet fever at six years of age, was this the immediate or remote result of it? No one living can give any more information than the patient himself, and so we will have to accept his statement as the one believed by his mother.

The question of the etiology of ophthalmoplegia externa is one of the greatest interest and has received constant and profound study everywhere. The origin of the 3d, 4th and 6th pairs of nerves has been the subject of investigation by many pathologists. The possible and probable damage to these nerves as they pass forward to enter the orbit from tumors, hæmorrhages, gumma, injuries, etc., has received much and well deserved attention. The literature on this subject is abundant and rich, especially in relation to the pathological cases—but not so much is to be found on cases like the above —when the extrinsic muscles are paralyzed and where the intrinsic muscles are intact.

One of the first cases to attract much attention by the profession occurred in the Eye Clinic of Prof. von Graefe, in Berlin, in 1856.

It was first published in *Graefe's Archives*, Vol. II, p. 299, as a case of Complete Paralysis of the Ocular Muscles and Ptosis of Both Upper Lids.

It occurred in a man forty years of age and was supposed to be due to an intra-cranial lesion, probably a tumor of the base of the brain. Both eyeballs were completely immobile, he having no voluntary power over his eyes in any direction. The eyes appeared prominent and were slightly divergent. He had suffered from diplopia but practically overcame this by using only one eye. There was marked paralysis of the left levator palpebræ superioris and there was a congenital coloboma iridis of the right side. The patient had complete accommodative power in both eyes and was able to read the finest print at the usual range. Tests were made by way of comparison between Graefe's, Liebreich's and the patient's eyes to prove this.

Graefe considered it at the time as a remarkable case of complete paralysis of all the extrinsic muscles with normal activity of the intrinsic muscles and it proved to him that the

activity of the ocular muscles was not a *sine qua non* condition for the integrity of the accommodative apparatus.

Since that time ophthalmology and cerebral pathology have made great advances and many points which were obscure are clearer now.

Gowers (Diseases of the Nervous System) says "that ophthalmoplegia externa is sometimes congenital and even hereditary. And he states that Hirschberg (*Berlin Gesellsch. Phys.*, June 8, 1886) has described a case of a man with congenital double ptosis and paralysis of all the ocular muscles, incomplete in the superior oblique and the internal muscles, whose mother presented a similar condition, while his own son had congenital ptosis and paralysis of the superior recti."

Noyes says that this disease "is is sometimes congenital, although some congenital defects of the eye muscles are due, not to paralysis, but to absence of muscles or their incorrect insertion or imperfect development."

Romano–Catania (*Bull. d'Ocul.*, xii, 21) reports a case of congenital ophthalmoplegia externa of both sides with the exception of both superior obliques. The patient had M. 9 D. R., and 10 D. L.

Möbius relates in the *Klin. Monatsbl. f. Augenheilk.* Vol. xvii, a case sf congenital atrophy of the ocular muscles.

The disease is occasionally seen when there is no well known cause to be assigned for it. Blocq (*Archiv. de Med. Experimen. Janvier*) saw a case of complete ophthalmoplegia in a woman about thirty years of age, in whom there could be discovered no etiological cause. The pupils reacted somewhat feebly to light but the accommodation was active.

While the disease is frequently progressive and perhaps generally so, yet it sometimes comes to a standstill.

Strümpel in his "Practice" says he has met with a patient with total bilateral ophthalmoplegia externa in whom this condition had existed without the slightest change for fifteen years.

Rumschewitsch (*Wjestnik. Ophth.*, 1883, No. 3) presents a compilation of 119 cases of ophthalmoplegia externa and says it may exist for years without complications. He states that in the majority of cases the sphincter iridis and ciliary muscle are not affected.

Uhthoff (*Tagebl. d. 59 Vers. deutsch. N. und Aertze in Ber-*

lin, p. 153) describes a case of a patient fifteen years of age, who had bilateral ophthalmoplegia externa and partial facial paralysis. There were no other cerebral complications.

The association of this disease with congenital defects in the development of the hands and feet has been observed by Gazepy.

In the *Arch. d'Oph.*, xiv, p. 273, he reports two remarkable cases. One that of a man, twenty-five years of age, who had congenital smallness of the index and little fingers of both hands and a webbed condition of both feet. He had double ptosis with paralysis of the superior recti and externi and with lagophthalmos. In addition to this there was in the left eye paralysis of the rectus inferior.

The other case was that of a girl, eighteen years of age, who had similar abnormalities of the hands and feet and ptosis of both sides. In the right eye there was paralysis of the rectus superior, inferior and internus; in the left eye paralysis of the rectus superior and externus with lagophthalmos.

Buch, in *Centralbl. f. Nervenheilk. und Psych.*, 1893, p. 57, reports a case of congenital double incomplete ptosis with a slight degree of right-sided divergent strabismus. Motions in all directions were very much limited. That the disease may be progressive after many years, is shown by the following: Hanke, in *Wien. klin. Wochensch.*, ii, 46, gives in detail the history of a woman, twenty-six years of age, with incomplete total ophthalmoplegia externa. Latterly there developed a paresis of the left and then of the right levator and almost at the same time a paralysis of the facial nerve.

Ophthalmoplegia externa completa is due to nuclear disease. Gowers says: "Paralysis of all the muscles of both eyes, internal and external, while theoretically conceivable from disease at the neighborhood of the orbital fissure and the optic foramen on each side, either in the orbit or within the skull, is practically met with in cases of nuclear disease."

No attempt has been made in this paper to mention the various phases of this disease which occur later in life, but only to record a few of the more interesting congenital cases. The cases are very various and cover a wide range of diseases.

Dalechow, in the *Zeitschr. f. klin. Mediz.*, xxii, reviews the literature on the subject and gives the following classification of diseases: They may be from, first, hæmorrhage, embolism

or thrombosis; second, syphilis, growth and softening of gumma: the origin can be central, nuclear or peripheral; third, from tuberculosis, rheumatism, influenza, meningitis, pneumonia, scarlet fever; from poisons: alcohol, nicotine and sausage; from tabes, multiple sclerosis, diabetes, Basedow's disease, gout, tumors, aneurisms and injuries.

A CASE OF RETINITIS PIGMENTOSA ASSOCIATED WITH CONGENITAL DEAF-MUTISM.

By HOWARD F. HANSELL, A.M., M.D.,

OF PHILADELPHIA,

Clinical Professor of Ophthalmology, Jefferson Medical College; Professor of Diseases of the Eye, Philadelphia Polyclinic, Etc.

It is with regret that we record our total ignorance of the etiology of acquired pigment-degeneration of the retina associated with optic nerve atrophy. The disease has mainly been ascribed to two causes, namely, inherited syphilis and consanguinity of the parents. In 4 out of a total of 44 cases observed by Leber (Graefe and Saemisch) no other cause could be assigned than a suspicion of syphilis; in 12 other cases there was a history of blood relationship of the parents; in 19 cases no cause could be named. Mooren and Nolden (quoted by Leber) attributed 25%, Fuchs 33%, to consanguinity It will be seen from these quotations that intermarriage among kindred has much to do with the causation of this disease. But we are forced to limit ourselves to this general statement. Further research does not enlighten us as to why the offspring of such marriages are thus afflicted. We know, however, that they are occasionally subject to defects that are congenital, such as hare-lip, idiocy, supernumerary fingers, deaf-mutism, etc., in conjunction with retinitis pigmentosa and have thus some ground for believing that the ocular condition is also congenital, although the latter does not develop until many years after birth. Some authors assert that retinitis pigmentosa is always congenital, notwithstanding that its symptoms do not appear in infancy nor can its ophthalmoscopic signs be detected at that period of life, but that pathologic changes in

the retina and nerve have commenced at or before birth, and, advancing by extremely slow stages, become manifest years later.

The causal relationship of syphilis, hereditary or acquired, is even more obscure, although reasoning from analogy, we have little doubt that this is an important factor. Interstitial keratitis, spinal sclerosis, and optic nerve atrophy are often due to the insidious action of the syphilitic poison. They appear in early adult life, are slow in growth and lead to unalterable tissue change; and yet their connection with syphilis, and why the poison waits for years to show itself as a cause for organic lesions or why one child of a family is afflicted while others escape, is an unsolved problem.

The following case, briefly described, throws absolutely no light on the question of etiology and yet I believe from its negative qualities it has a certain value: B. S., a strong, muscular, well-built man of 33 years, the eldest of a family of several children, was born a deaf-mute. At 15 years of age, he commenced to see indistinctly at night and at other times, when the illumination was bad. The field of vision gradually grew less until at the time of my examination it included only the foveal region, and the acuity was reduced to perception of light only. Until a few months ago he was able to do the rough work on a farm and now is obliged to seek refuge in a blind asylum. He had learned the sign language, and formerly could converse freely with the members of the family, but lately, no longer able to see the signs, those who would talk with him must form the letters on some portion of his body, usually the hands, so that the characters could be felt. Thus he was able to understand as perfectly and nearly as quickly as formerly. There was no family history of consanguineous marriages among parents and ancestors. His father and mother were not even distantly connected. His brothers and sisters had no birth-marks, congenital defects, or other abnormalities. Moreover, there was no history of syphilis, congenital, inherited, or acquired. His mental faculties were in no wise clouded, and his physical strength and endurance were phenomenal. The ophthalmoscopic signs were typical of retinitis pigmentosa. The pigment patches were abundant throughout the entire fundus even up to the edge of the disc. The nerve-head was perfectly white and traversed by few

bloodvessels which could be followed but a short distance from the edge of the disc when they were changed into white lines. Both eyes were involved, as is always the case, and no difference in the degree of the degeneration could be detected. The pupils were moderately dilated and responded well to the influence of light and associated third nerve stimulus.

My information as to the history of the case was furnished by a brother, himself a large man and healthy in every respect. He assured me positively that there was no hereditary disease in the family; and that his parents were hard-working people, and that the case just recorded was the only exception as far as he knew to an extremely healthy family. Accepting his statements, confirmed by my own limited observation, as true, I am obliged to confess that I am entirely in the dark as to the cause of the disease in this unfortunate man.

REPORT OF A CASE OF MIND-BLINDNESS DUE TO CEREBRAL COMPRESSION.

By L. R. CULBERTSON, M.D.,

OF ZANESVILLE, OHIO.

Mr. L. M. Johnson, age 30 years, news agent. Was sent to me by his attending physician, Dr. C. M. Rambo, of this city. I did not test the visual field nor the pupillary reaction to light. The ophthalmoscopic examination showed: R. E., severe optic neuritis; inner half of disc obscured and outer half cupped and grey in color, the veins greatly enlarged and arteries small. L. E., the same condition as in the right eye, save that the neuritis is not so severe. Patient says he has pain in the back of his head. I diagnosed either glioma of the left cortex in parieto-occipital region; or circumscribed meningitis with adhesion in same region. Dr. Rambo put him on heavy doses of bromide and iodide of potassium, and later enormous doses of mercury and iodide without producing any benefit. Later he was under the treatment of Dr. Starling Loving, of Columbus, Ohio, who gave him the same treatment, but without benefit. He gradually grew worse and the pain became violent and constant, so that he could not sleep.

A month before his death acute meningitis developed. At

this time there was a spot on the left parietal bone corresponding to the convolution of the angular gyrus, at which he had constant pain and when pressure was exerted at this point, the pain was increased. The consulting surgeons, Drs. Rambo, Logsdon, and myself, advised trephining, but his wife was opposed to this. About this time when he would look at his friends and someone would ask him if he knew a certain person, he would say no; but if the person referred to would speak to him he would say yes and give his name. Yet if the same person saw him next day he did not remember him [1]

We made no test as to hemiachromatopsia, nor word-blindness, nor agraphia, nor hemianopsia. There was no epilepsy or epileptiform seizures nor paralysis of motion or sensation. A few weeks before death he became delirious and soon after, from compression, developed paralysis of deglutition, etc.

Post-Mortem Examination.

Very thick calvarium. Dura-mater normal on right side; very greatly thickened on left side. Arachnoid greatly thickened on left side and vessels enormously enlarged. Enormous increase in sub-arachnoidal fluid. Sylvian vessels greatly enlarged on left side. Ascending parietal branches of sylvian vessels enormously enlarged. Considerable softening of occipito-temporal (left side) convolution and slight softening of angular gyrus and supra-marginal convolutions (left side). No softening at any other points either on cortex or at base, excepting that there was considerable softening of the optic tracts and nerves. Cerebellum normal. There was a deep depression in the frontal bone large enough to sink one's thumb in to a depth of one-eighth of an inch, and the inner table was depressed this much, but the brain tissue was perfectly normal at this point. This injury was received when he was a child. On inspection of the inner surface of the left parietal bone we found necrosis of the inner table covering a surface almost two inches square. At one point—corresponding to the seat of pain and tenderness on pressure—a surface as large as a quarter was so necrosed as to be translucent This point lay over the occipito-parietal and supra-marginal convolutions. Bloodvessels normal in texture.

[1] See Bowman Lecture, 1888, for similar case.

REMARKS.

In the history of this case there was nothing specific. No sunstroke. About a year ago he was struck on the back of his head while exercising in a gymnasium. Our belief is that this was the cause of the disease, and that it produced circumscribed meningitis and osteitis.

He had some aphasia before he became unconscious, but this was a distant symptom as the autopsy showed that the speech center was not softened.

This case would tend to substantiate the opinion of Swanzy[2] that the center for visual memory lies in the parieto-occipital lobe. The angular gyrus and supra-marginal convolution lie so close to this that it would have been impossible to determine which convolution was primarily affected, were it not for the fact that there was far greater softening of the parieto-occipital than of the angular gyrus and supra-marginal convolution. The occipital convolutions were not softened. It is to be greatly regretted that the pupil reflexes and visual fields were not tested sooner. When the pupil reflexes were examined—late in the disease—they did not respond to light in any part of the field.

EDITORIAL NOTES.

The reproductions of the photographs to my article in the February number, have been so absolutely unsatisfactory, that I have had them printed again on special paper enclosed in this number of the JOURNAL.

As the former publishers of this JOURNAL have kept no stock of extra numbers, it is impossible for me to fill all the many demands for missing numbers of the JOURNAL. If any of our readers could sell us the December number of 1894, February, March, August and September numbers of 1895, we should be very glad, as these numbers in particular do not seem to have reached some subscribers. Although I can not consider myself responsible for the shortcomings of the former publishers, I like, as much as is in my power, to make our subscribers satisfied.

ALT.

[2] Transactions of the Ophthalmological Society of the United Kingdom, 1888.

CLINICAL MEMORANDA.

A RARE FORM OF RETINO-CHOROIDAL DEGENERATION.

By J. ELLIS JENNINGS, M.D.,

OF ST. LOUIS, MO.

The following rare case of retino-choroidal degeneration was seen at Prof. Alt's Clinic January 31, 1896. It closely resembles three cases from the Clinic of Prof. Fuchs, of Vienna, reported by Dr. Colman W. Cutter in the *Archives of Ophthalmology*, July, 1895.

Mr. B., aged 49, has had poor vision since childhood, but managed to get along fairly well until about fifteen years ago, when he suffered from some inflammatory disease of the eyes

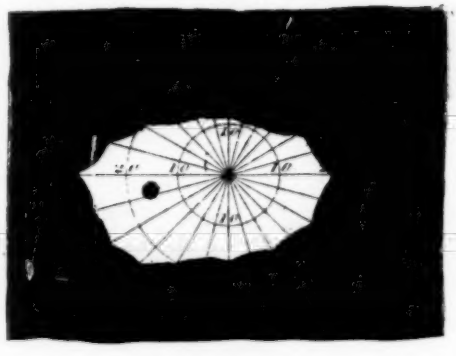

FIG. 1.

accompanied by severe pain and headache. He dates his bad sight from this attack and states that it gradually got worse until five years ago he was forced to give up his position in consequence.

He also had difficulty in finding his way about at night (nyctalopia). No specific history, inherited or acquired, was obtained. His father, mother, brother and one sister had good

eyes. The other sister, since dead, had bad sight which the patient imagines was much the same as his own.

Vision, O. D. $^{2}/_{cc}$; O. S. $^{3}/_{cc}$; not improved by glasses.

The refractive error is about — 8.00 D.

The field of vision in both eyes is very much narrowed concentrically. Fig. 1 shows the limits for white in the left eye. The field for blue, yellow, red and green lie just within the white zone, in fact all colors are recognized almost as soon as white. In both eyes there is a posterior cortical opacity, quite dense centrally but still allowing a fairly good view of the fundus to be obtained. The ophthalmoscopic picture is peculiar and is alike in both eyes.

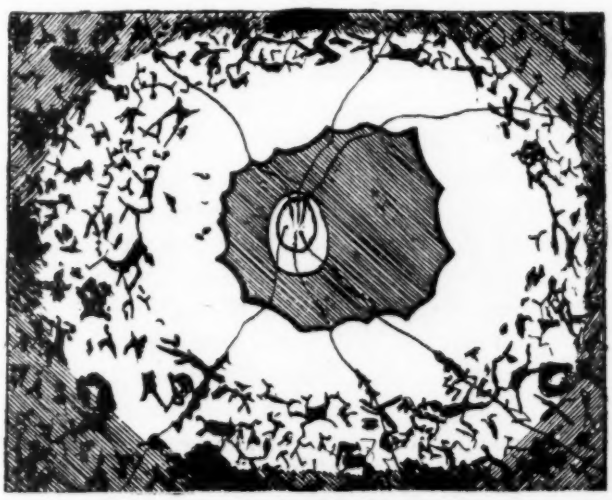

FIG. 2.

Fig. 2 is a drawing of the left fundus by the direct method. The optic nerve is pale and is surrounded by a rather large staphyloma. The vessels are narrow and with difficulty traced peripherally. Surrounding the disc and staphyloma is a normal zone of fundus which terminates abruptly in a glistening white zone of choroidal atrophy with clean-cut, scolloped edges. Beyond the white zone the fundus gradually colors into the normal hue showing here and there a few choroidal vessels. The diagonal lines in the drawing denote normal fundus. The atrophic zone and peripheral portions of the eye

ground are thickly covered with a fantastic meshwork of pigment which usually assumes the bone corpuscle shape so characteristic of retinitis pigmentosa. But in places are seen round or irregular blotches such as we find in choroiditis. If it is true, as some authors state, that atrophic changes are never seen in retinitis pigmentosa, then the case is evidently a rare form of retino-choroidal degeneration with pigment deposits closely resembling those found in retinitis pigmentosa.

FOREIGN BODY IN ORBIT: THE BILL OF A FISH.

By W. BURRELL THOMSON,

Surgeon-Major, A.M.S., York.

Lance-Corporal G. S., 2nd Battalion West Riding Regiment, while at a bathing parade in Barbadoes, W, I., on July 21, 1891, was struck by a fish. About half an hour later, on admission into hospital, he presented a small lacerated wound beneath the right orbital ridge, at the junction of its outer and middle thirds. On examination with the finger a rough body was felt embedded in the orbit. This required some amount of force for its removal, having apparently passed from the outer side downward and inward behind the eyeball, to be wedged into the bones on the inner side of the orbit. Some slight bleeding occurred from the right nostril on its removal. This foreign body was found to be a bill of a fish, two and a half inches in length; the greatest width was one-third inch, and it was armed with six teeth, the longest of which was one-seventh of an inch. The eyeball, which was intact, had been levered forward; it protruded considerably and was fixed and immovable. The conjunctiva on the edge of the globe was torn transversely. There was some ptosis. The pupil was widely dilated and reacted slowly. There was no vision except perception of light. The man was subsequently invalided from the service with loss of vision from atrophy of the right optic disc. Surgeon Major Whitehead, A.M.S, Assistant Professor of Surgery, reports that on his admission into Netley on April 18, 1892, there was found to be "marked atrophy of the right optic disc."—*British Medical Journal.*

OPHTHALMIC DIGEST.

By J. ELLIS JENNINGS, M.D.,
OF ST LOUIS, MO.

SEROUS CYCLITIS.

N. C. RIDLEY. ("Royal London Hospital Reports," Vol. XIV, Part 1, 1895) says:

This disease is known also by the names serous iridocyclitis or serous uveitis, according as the anterior part or the whole of the uveal tract may have been involved, and formerly was frequently termed descemetitis or keratitis punctata, from the presence of dotted opacities seen when light is transmitted through the cornea.

CAUSES.—The following is a list of causes of the disease given by authors: 1. Syphilis. 2. Sympathetic trouble after injury to the other aye. 3. Traumatism 4. Gonorrhœa. 5. Uterine disease. 6. Rheumatism. 7. Gout. 8. Diabetes. 9. Intra-ocular growths involving the ciliary region, including tubercle. 10. Spreading of inflammation from neighboring parts. 11. Following acute specific fevers. Besides those due to the above causes, many cases occur in which no cause can be found, and these are called idiopathic.

SYMPTOMS.—In spite of the wide difference of etiology, most of the cases present many points in common in their clinical history and course, and also in histology.

In all the well-marked cases there was great injection of the episcleral vessels, especially at the sclero-corneal junction, accompanied by more or less pain, ciliary hyperæsthesia, and loss of vision. On examination, the media were hazy and the vitreous was seen to contain floating opacities. The iris escaped in most of the cases, but when involved, exhibited the ordinary signs of iritis, viz., alteration of color, dulness of lustre, sluggish reaction, and contracted pupil. In some instances a total or partial paralysis of the ciliary muscle was present, and in others slight photophobia and lachrymation.

On examination with focal illumination a number of

grayish dots were noticeable on the posterior surface of the cornea arranged roughly in the form of a triangle; with the apex at the centre and base downwards, but frequently dis tributed over the whole of the surface, and when large looking like round specks of grease. All the cases which continued under obversation for some time were subject to occasional exacerbations of pain, ciliary tenderness, and redness, with a tendency to increased intra-ocular tension, the latter sometimes rising to T. + 1 or + 2. Those with high intra-ocular tension could be divided into two groups, in one of which there was an anterior chamber shallower than usual, with dilated pupil as in primary glaucoma, and the second in which the anterior chamber was increased in depth, with a pupil of about the normal size. The decrease of vision was attributable in the earlier stages to the haziness of the media, but subsequently to the increased tension and to accompanying choroido-retinal changes.

Examination of Eyes After Removal.

Group I.—Here the anterior chamber is shallow, lens and iris pushed forwards, and the pupil is dilated. In most cases the iris is adherent to the cornea for a greater or less distance at the periphery. In the earlier stage of the disease the circumlental space is somewhat diminished by swelling of the ciliary processes, but later on it is again increased, owing to the atrophy of the processes from the rise of intra-ocular tension. The aqueous and vitreous chambers contain an albuminous fluid which is coagulated by the ordinary hardening reagents, and can be recognized from the fact of its taking a deeper stain with eosin than does the celloidin in which the specimen is imbedded.

On microscopic examination, the swelling of the ciliary body and processes is seen to be due to injection of the vessels, with exudation of lymph and leucocytes. There are also great proliferation of the epithelial cells, not only of the pars ciliaris retinal, but of the hexagonal pigment layer beneath, many of them being shed and, together with leucocytes, found loose in the vitreous chamber. The dulness of the media and opacities in the vitreous observed clinically are, no doubt, due to these, together with the exuded lymph. On looking at the suspensory ligament it can be seen that many of these epithe-

lial cells, and some of the leucocytes, having been carried forward with the lymph stream, have become entangled in the meshwork between the fibres constituting that structure. In consequence, additional obstruction has been caused to the exit of the intra-ocular fluid from the vitreous into the posterior chamber, through the already diminished circumlental space. The tention in the vitreous chamber has then evidently risen, and the lens been pushed forward, and subsequent events have taken place as in ordinary primary galucoma.

Group II.—In this group the chief cause which leads to all the other anatomical differences is, that there is either very little or no proliferation of the epithelium on the ciliary process. Consequently the only solid bodies thrown into the lymph stream through the vitreous are leucocytes, and these can readily pass through the meshes of the suspensory ligament under ordinary conditions. These and other inflammatory products reach the posterior chamber, and are carried thence through the pupil into the anterior chamber. Here many of them being washed against the posterior surface of the cornea, and being more or less sticky, either from their own natural property or from the accompanying albuminous exudation, adhere, and on having become attached, others are attracted to adhere to it, and so little isolated heaps are formed and not a continuous sheet. These give rise to the dotted opacities on the cornea. By far the greater number of the leucocytes, however, are carried on in the lymph stream towards the angle of the anterior chamber, and here some of them may pass through the spaces of Fontana in the ligamentum pectinatum, and so get removed. But many of them do not escape, but becoming entangled in the filter, whose meshes are much finer than those of the suspensory ligament, cause a gradually increasing obstruction, accompanied by inflammation in that neighborhood. The primary obstruction occurring here at the outlet of the anterior chamber, pressure is exerted on all sides of that cavity, and so the iris and lens are thrust backwards, the angle is widened, and the whole chamber increased in depth Pigmented cells may occasionally be found in the deposits on the back of the cornea, but they are never abundant, since in this group of cases there is very little proliferation of epithelium. In Group I, where the primary obstruction is at the suspensory ligament, leucocytes

can at first pass through that structure, and some of them adhering to the back of the cornea give rise to small masses just as in the second; these masses are never so large as those which occur in the latter, where the primary obstruction is in the spaces of Fontana, and where the anterior chamber is deep.

In the examination of an eye that has been blind for some time from secondary glaucoma of the kind spoken of in Group I, it is at first rather difficult to trace the sequence of events, as the lens and suspensory ligament are frequently in their normal situation. This I consider to be due to the following circumstances: The lens and iris are at first pushed forward, and the iris becomes congested and adheres to the cornea at its periphery, obliterating the angle of the anterior chamber, thus shifting the locus of the obstruction more forward. After the tension has been raised for some time, atrophy of the ciliary processes takes place, and the circumlental space is increased, and so the causes working at the original site of obstruction are diminished. In consequence the accumulation of pressure occurs as in Group II, in the anterior chamber; but the adhesion of the iris to the cornea prevents the former structure being thrust back again and the anterior chamber deepened, therefore the lens is being pushed back by itself to its original situation, and we have as a result, a deep posterior and a shallow anterior chamber. In other instances the anterior chamber becomes deepened, although the iris is still adherent at the extreme periphery, and the angle effectually occluded. The deepening of the posterior chamber and recession of the lens are also conduced to by the diminution of secretion following atrophy of the ciliary processes and compression of the retinal vessels and consequent lowering of tension in the vitreous chamber. Though some authors distinguish between the cyclitis which is serous, and that in which the exudation is plastic, no distinct line can be drawn, as in all cases in which the disease has persisted for a time the exuded fluid becomes plastic, even though it were partly serous at first. It is owing to the construction of this plastic material that another alteration in the anterior chamber occurs in old standing cases of Group II. This newly organizing tissue is chiefly found in the circumlental space, and so on contracting draws the root of the iris backwards, and laterally widens the an-

Plates to Dr. Alt's paper in the February No., to replace the unsuccessful illustrations.

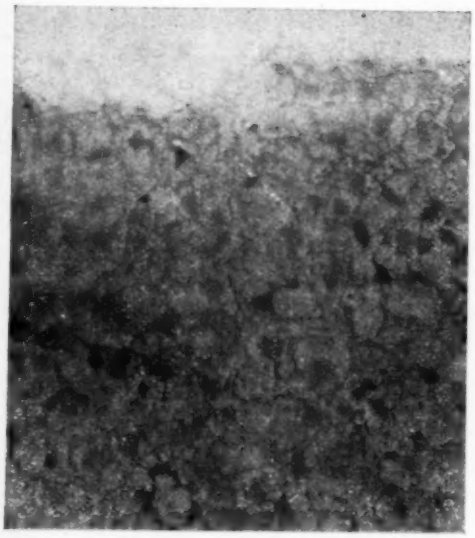

FIG. 1.

FIG 3.

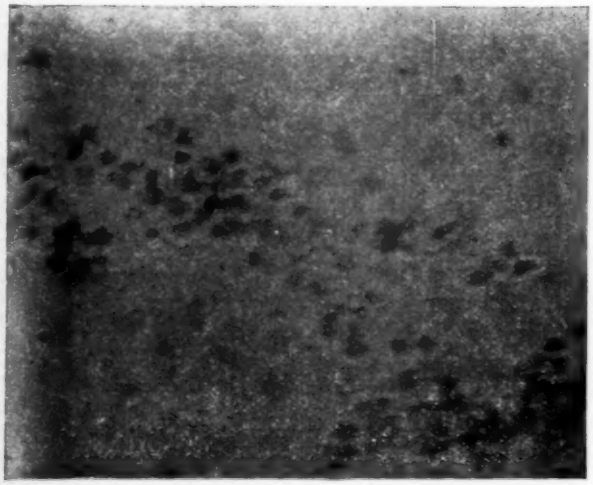

FIG. 4.

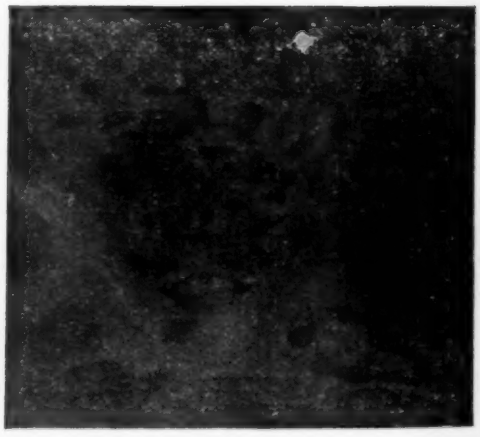

FIG 7.

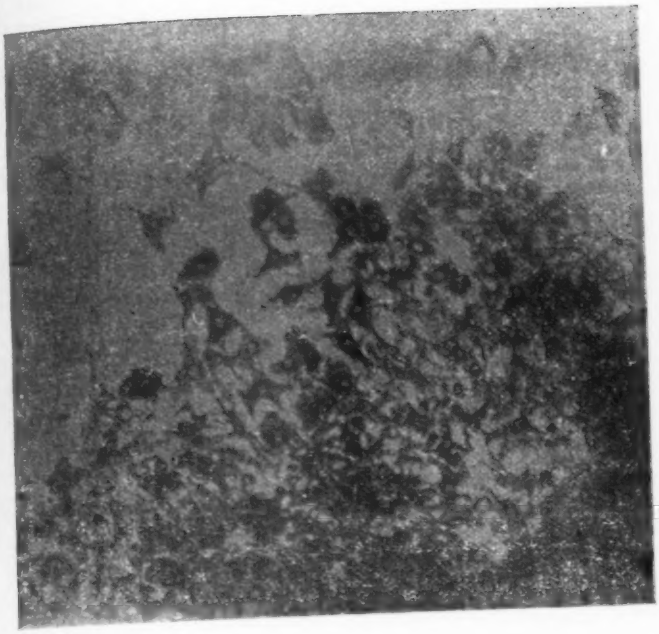

FIG. 5.

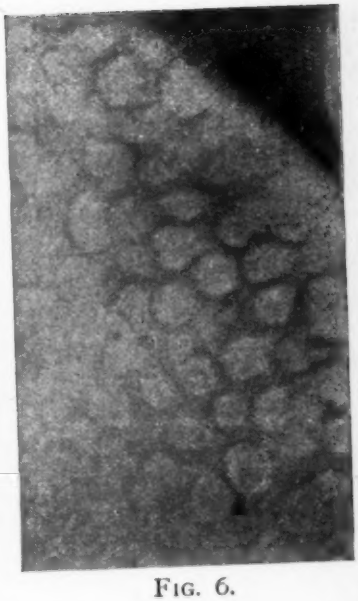

FIG. 6.

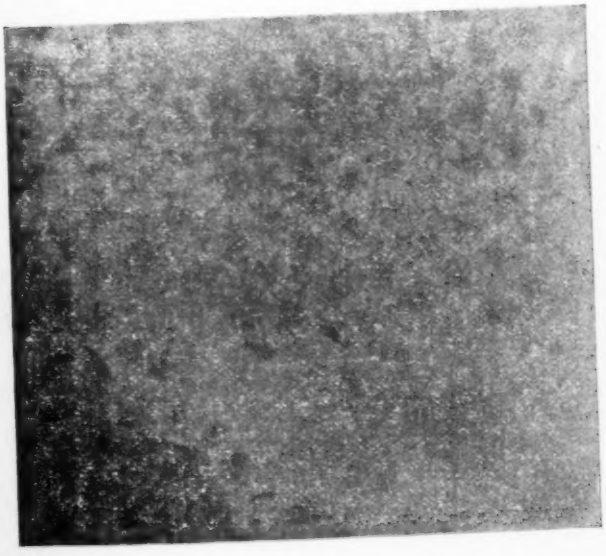

FIG. 8.
Similar Condition as in Fig. 2.

FIG. 2.

terior chamber; and being attached to the periphery of the retina behind, detaches that structure in a circular fold at the ora serrata. With regard to the collection of leucocytes and lymph on the posterior surface of the cornea, changes may occur in the cubical endothelium between them and Descemet's membrane, but it is not the rule, and the layer is normal at the commencement of the deposit, and is still healthy if the deposit be early washed away, and the cornea may even in the most marked cases become eventually quite clear.

TREATMENT.

Iridectomy.—From a consideration of the causes of obstruction it will be obvious that, except perhaps now and then in cases of Group I, removal of a larger or smaller segment of the iris in the operation of iridectomy would be of no avail, either to stay the process of inflammation or to permanently reduce the intra-ocular tension; and in fact in consequence of the organization of inflammatory products near the iridocorneal angle in the process of repair more obstruction may be caused and the same condition rendered worse. Another danger after iridectomy is from the blocking of the spaces of Fontana by pigmented epithelial cells of the iris which may proliferate at the site of section and be set free.

Paracentesis.—It seems reasonable to suppose that the reduction of tension can best be effected by repeated paracentesis of the anterior chamber, so evacuating inflammatory products with the intra-ocular fluid. Probably the paracentesis should be done in front of, rather than in the sclero-corneal junction, so avoiding the extension of the inflammation at the site of the puncture to a part already damaged.

Eserine.—In some cases, the use of eserine is indicated, as for instance in the early stage of those in which the original obstruction is in the circumlental space; but it should not be persisted in if there be signs of the inflammatory process spreading to the iris, and it must be remembered that this drug produces congestion of the ciliary processes.

Mydriatics.—In the cases in which the anterior chamber is deep, a weak mydriatic (atropine gr. $^1/_4$ to ℥j., or even less) may be circumspectly used, a careful watch being kept upon the tension. The objects of the treatment being to paralyze

the ciliary muscle and to dilate the pupil and prevent the formation of posterior synechiæ. I have seen exclusion of the pupil and "iris bombé" result in one case in which this had not been done.

Essentials in Treatment.—The main essentials in treatment are to give as complete rest as possible, *e. g.*, dark glasses, and the avoidance of near work, combined with attention to any general disease concerned in the causation.

Prognosis.—The prognosis, unfortunately, at present is bad, owing to the great tendency to recurrence, and in nearly every case the vision is permanently damaged to a greater or less degree.

THE IRIS AS A DIAPHRAGM AND PHOTOSTAT.

CHAS. F. PRENTICE, (*Annals of Ophthalmology and Otology*, Vol. IV, No. 4):

Under this title it is proposed to inquire into the value of sub-decimals of the diopter-lens in ametropia. In every compound lenticular system we are met with the necessity of providing against spherical aberration. This is accomplished in the construction of optical instruments, by introducing a diaphragm between the lenses to exclude peripheral rays. If the proper diaphragm be replaced by one of smaller aperture, we increase the definition, but diminish the extent of field and illumination and *vice versa*. The aperture of the diaphragm must therefore have a definite diameter for every optical instrument, if we are to secure *maximum* definition and illumination, *without aberration*. The proper diaphragm is therefore one of the most important and indispensable parts of every compound dioptric system. The human eye is such a system, and is provided with its diaphragm—the iris. In the eye, which is a dynamic apparatus given to variations of power, a fixed diameter of pupil would fail to theoretically fulfill the requirements. When the eye is in a state of accommodation, it becomes a stronger refracting system, and therefore needs a smaller aperture of diaphragm; hence the pupil contracts. It is universally admitted that the iris acts independently of, and

simultaneously with accommodation. When acting independently of accommodation, the iris is a highly sensitive photostat, regulating the volume of light upon the retina to such a degree as shall be most agreeable to our light-perceptive sense. A most suitable and synchronous balance, between retinal perception, uveal stimulus, and iritic respose, must therefore exist, if the iris is to perform its functions simultaneously as diaphragm and photostat. An endeavor will here be made to support the hypothesis that *a disturbed equilibrium of these functions is probably the cause of asthenopia in low degrees of ametropia.*

We have thus far been content to know that pupils differ in size in different persons. There must, however, be a limit to the maximum diameter of the pupil, if aberration is to be excluded, and if, for any reason, the pupil is prevented from contracting to at least this limit, we shall have aberration even in the emmetropic, and which to all practical purposes, would be equally as effective in impairing vision as a low degree of myopia. This is undoubtedly one reason why errors of refraction of the same degree are not accompanied by the same diminution of visual acuteness. The myope of 1 D., with small pupils, *without* glasses, will probably have better vision than the myope of ,1 D. with much larger pupils. Within certain limits, peripheral aberration and anomalies of refraction are analagous in destroying definition of the image. A slight error of refraction, with large pupils, may produce diffusion images equally as pronounced as considerable refractive error with small pupils. *Asthenopia is therefore quite as apt to be experienced on account of the size of the pupil, as it is on account of the error of refraction.* This should explain why it is that many persons, having small pupils, endure a considerable error of refraction without inconvenience, while others, with large pupils and small errors of refraction, are afflicted with asthenopia.

So far, we have no means of ascertaining the size, or that variation of the pupil which is necessary to establish the proper harmony between refraction, accommodation, illumination and freedom from aberration. The intuitive discrimination, which accompanies experience, is at present our only guide.

In refractive errors of low degree, which are relieved by

lenticular correction, the retinal perception is usually also very keen, thus increasing stimulus to contraction of the sphincter, while the correction in such cases frequently improves vision to $^{6}/_{3}$, which is far above normal.

The larger the pupil, the more pronounced will be improvement in visual acuteness obtained by low-degree corrections. The quarter-diopter lens rarely proves of benefit when the pupils are small.

Again, patients frequently wear such glasses for a time, relieving their asthenopia, and ultimately lay them aside, without feeling the necessity of their further use. Examination will nevertheless reveal the fact that the *optical error has not changed.* Closer examination, however, will frequently show that the pupils appear to be smaller at the time the patient has discarded his glasses, than when they were prescribed. The pupil being the only member seeming to have undergone a change, are we not justified in suspecting the iris, by reason of disturbed innervation, as having been at least implicated in the cause of asthenopia?

CONTRIBUTIONS TO THE KNOWLEDGE OF THE HISTOLOGICAL ALTERATIONS IN THE RETINA AFTER EXPERIMENTAL INJURIES.

DR. A. SEPLJASCHIN, (*Archives of Ophthalmology*, Vol. XXIV, No. 4):

The experiments were made on rabbits in the following manner: After pulling the eye either inwards or downwards with a pair of forceps, a discission-needle is plunged through the wall of the eye, five or six mm. from the edge of the cornea, avoiding injury to the lens. The needle is carefully run through the vitreous aud by appropriate movement of the handle a cut is made in the retina. The eyes were enucleated at periods of from 22 hours to 285 days in a total of about 50 experiments.

The data obtained by our investigations show that after injury of the retina an inflammatory process begins in the region of the injury in which the retinal elements undergo

changes of both a progressive and a regressive character. Changes of the latter character are found at the point of most severe inflammation, both in the nervous and the visual elements. The progressive changes in the beginning are limited to regenerative processes, but a regeneration of nervous and visual elements that have been destroyed does not occur. The supporting framework, however, proliferates, but this never leads to union of the margins of the wound. There takes place an atrophy of the retina and the chorid in the region of the retinal wound, due to serous chorio-retinitis, after which the connective-tissue elements of the retina and choroid form a scar. In penetrating wounds the gap is closed mostly by proliferating epibulbar connective tissue, less by proliferation of choroidal tissue, and probably in small measure also by the wandering cells of the vitreous which become fixed connective tissue cells.

DIPLOPIA IN THE PERIPHERY OF THE FIELD OF FIXATION AND ITS BEARING ON THE DIAGNOSIS OF MUSCULAR PARALYSIS.

DR. A. DUANE, (*Archives of Ophthalmology*, Vol. XXIV, No. 4):

Dr. Duane's conclusions drawn from a large number of examinations of the field of fixation and of diplopia occurring in all parts of the field, are as follows:

1. Diplopia occurring at the limits of the field of fixation (beyond 45° from the primary position) is physiological. It is not, however, by any means always present even there, and in any case is slight in amount, and in the same case often variable and inconstant. It may be due to a natural failure of one eye to keep up with the other in its excursion, or to the effects of projection, being in the latter case brought about by the rotation of the retinal horizon, real or apparent, that occurs in oblique positions of the gaze. But whether projection can produce any diplopia at all of the kind that I found in my cases is doubtful, and in any case the effect of projection in

producing vertical diplopia is very slight and makes itself apparent only at the limits of the field of fixation.

2. In a large number (probably the majority) of persons with normal eyes it can be proved that binocular single vision is still present even when the eyes are carried further than 45° from the primary position.

3. Well-marked diplopia occurring as a constant phenomenon within 40° of the primary position indicates a weakness or at least a want of balance of the eye-muscles. If this diplopia is still quite peripherally located, *i. e.*, occurs not less than 30° from the primary position, it indicates only a slight and, very likely, temporary muscular disability, such as may occur in neurasthenia, etc. As weakness from this cause is very apt to be symmetrical, the diplopia is often present to about an equal extent in all the oblique positions of the gaze, both to the right and left, and above and below.

4. A diplopia which begins to be apparent near the primary position and increases rapidly as the eyes are carried in any one direction, indicates a serious impairment of muscular energy, *i. e.*, a true paresis. The diagnosis will be confirmed if upon repeated examinations it is found to be constantly present and particularly if it shows a markedly unilateral character.

A CLINICAL AND EXPERIMENTAL STUDY OF THE SO-CALLED OYSTER SHUCKER'S KERATITIS.

R. L. RANDOLPH, M.D., (*Johns Hopkins Hospital Bulletin*, November–December, 1895):

Oyster shucker's keratitis may be defined as a traumatic keratitis where the injury is produced by a particle of the oyster shell. A minute particle of the shell is violently chipped off by the hammer that is used in the shucking process and flies into the eye. The particle is generally too small and too light to penetrate to any distance into the cornea. Large pieces, however, are sometimes detached and are driven through the entire thickness of the cornea, and when such a thing happens, happily rare, loss of the eye usually results.

Symptoms—The photophobia is marked and there is a defined sensation of having been struck in the eye. This sensation is not usually followed by pain until some hours later. Frequently the exposure to artificial light in the evening of the same day will mark the time when the unpleasant symptoms begin. From now on the pain is usually intense, and the clinical symptoms resemble those of phlyctenular keratitis somewhat intensified. The disease is chiefly remarkable for the rapidity with which the cornea undergoes necrosis at the site of the injury, this area of necrosis being usually very small, owing no doubt to the small size of the foreign body. Small foreign bodies of copper, steel and sand usually produce no appreciable keratitis; and even when they lodge in the cornea, commonly require several days to cause a noticeable inflammation. On the other hand, the oyster shucker presents a marked infiltration at the point of injury within twenty-four hours after the accident. This decided reaction on the part of the cornea makes the injury a peculiarly dangerous one when a large area is wounded, these conditions being invariably followed by loss of the eye through panophthalmitis.

Bacteriological investigations failed to discover any specific organisms, which manifested any pathogenic properties when introduced into the corneæ of rabbits. The carbonate of lime, of which the oyster shell is almost entirely composed, was found to possess qualities irritating enough to call forth a keratitis when introduced into the cornea of a rabbit, and it is more than probable that several other chemical ingredients of the shell would be more or less irritating to the cornea.

It is certain that bacteria always play a part in traumatic keratitis, but it is evident that in this variety of traumatic keratitis the cornea is rendered especially susceptible to the effects of micro-organisms, by the irritating chemical ingredients of the oyster shell, notably the carbonate of lime.

Treatment.—The yellow salve has proved useless in our hands. The galvano-cautery was used in a certain number of cases, but it did not seem to exercise any specific influence for good, and the same can be said of eserine. A compress bandage and a mild sublimate solution ($^1/_{4000}$) used every four hours, together with an occasional drop of a solution of atropia—one per cent.—have given the best results. To this treatment the keratitis responds promptly, and in a week or ten days the

subjective phenomena have been so ameliorated that the shucker can resume work. The opacity can be detected by oblique illumination and is permanent.

BRACKET FOR TEST CARDS.

J. THORINGTON, M.D., (*The Philadelphia Polyclinic*, Nov. 9, 1895):

While the accompanying illustration is in great part self-

explanatory, it may be well to add that the bracket is made in two parts, *i. e.*, one small piece of polished oak board to each end of which is screwed a vertically placed brass socket; the other, a brass rod curved to a half circle and one inch of each end bent down to a right angle so as to fit evenly in the sockets on the board. From this description it will be seen that the bracket can be fastened to any convenient flat surface by means of screws passing through the oak. The cards are easily adjusted by raising one end of the rod out of the socket and slipping it through the round opening in the card.

Points of merit:

(1) Many cards may be hung one over the other.
(2) The cards are always in place.
(3) There is no taking down or hanging up.
(4) The cards do not become soiled, bent or broken.
(5) When it is desired to change cards, all that is necessary is to turn the upper card on the rod, toward the left.

Made by Wall & Ochs, Philadelphia.

WHICH NERVES GIVE RISE TO THE SENSATION OF PHOTOPHOBIA?

H. GRADLE, (*Annals of Ophthalmology and Otology*, Vol. IV, No. 4):

The following accidental observation proves that the sensory nerves of the cornea can be influenced by light under some circumstances, and that their irritation by light can cause photophobia:

Dr. B. became blind in the left eye during childhood, in consequence of a blow, causing white atrophy of the optic nerve. In October Dr. B. suffered of a circumscribed keratitis of the left eye, which under treatment rapidly improved. But in December there developed a small shallow ulcer, with gray floor, in the lower part of the still hazy cornea, which caused him more acute annoyance. During the time of acute irritation Dr. B. stated that the *blind eye was sensitive to light.* It presented the usual appearances of ciliary irritation, viz., ciliary injection, watering and partial closure of the upper lid. This partial ptosis, due no doubt to a reflex tonic contraction of the orbicularis muscle, was distinctly increased on exposing the eye to light, and lessened by relative darkness.

Moreover, Dr. B. *could tell promptly whenever I threw light into the blind eye* by means of a mirror in the dark room, the other eye being of course excluded. The sensation due to light was one of increased discomfort, not easily described in words, with a tendency to shut the eye. There was neither a sensation of light nor of warmth. After the disease had healed the eye was entirely unconscious of light thrown into it.

Conclusions.—1. The sensation of photophobia can be induced by sensory nerves without activity of the optic nerve.

2. That the nerve fibres involved are those of the cornea is most probable in view of the paucity of sensory fibres in the other ocular tissues and from the fact that photophobia occurs most frequently in diseases of the cornea.

3. The instillation of cocaine gives decided relief in photophobia by its action on the corneal nerves.

4. Our observation, however, does not prove that photophobia may not also be induced through the optic nerve. It is probable that the distress to which light can give rise in some diseases, and under some circumstances even in health is the result of an unusual activity of the optic nerve fibres.

THE OCULISTS' SIGHT TESTER.

This optometer, manufactured by the Optical Institute, London, 89, Hatton Garden, consists of a revolving mahogany disc, containing lenses from + or — 1 D. to 8 D., and of an extra metal revolving quadrant containing the fractions of dioptres, and + and — 8 D.; by combination of disc and quadrant lenses varying in strength from + or — 0.25 D. to + or — 16 D. can be obtained. By a single revolution of the disc and quadrant, these glasses can be rapidly passed in front of the eye. The apparatus can be used with the ordinary Snellen's types for distant vision; for near vision a test card on a movable arm is supplied with the instrument. For the use of spherical lenses only the optometer is simple, and handier than a test case; it will be found useful in the dark room in the application of the shadow test, as the change from one glass to another is made by a single revolution of the wheel. For the finer kind of refractive work, necessitating the use of cylinders, however, the instrument is not available; and the solid thickness of the disc and quadrant somewhat impair the accuracy of the results in lenses of high power, owing to the separation of the lenses from each other, and from the eye under observation The cost of the sight tester is £2, 12s. 6d. —*British Medical Journal.*

SOCIETY PROCEEDINGS.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

EDWARD NETTLESHIP, F.R.C.S., President, in the Chair.

THURSDAY, JANUARY 30, 1896.

THE VISUAL EFFECTS OF REFRACTIVE ERROR.

DR. GEO. J. BULL (Paris) read this paper. He gave an account of a series of experiments in which he had endeavored to reproduce with the photographic camera the effects of the different degrees of myopia, hypermetropia, and astigmia. The effect of a given error of refraction upon the photographic appearance of the test types was different in certain characteristic respects from the image seen by the eye. By further investigation he arrived at the conclusion that this difference was accounted for by the fact that there was in every case of refractive error an element of monocular diplopia, which he gave reasons for ascribing to the sectors of the crystalline lens. By adding to the photographic camera a lens which was itself divided into sectors he had been able to produce a photographic image of the test types closely analogous to the visual image with the corresponding error. A series of these photographs were presented at the meeting, and will appear in the *Transactions*.

THE PRESIDENT remarked that monocular diplopia was not a common complaint of patients with refractive error; if the phenomenon were constantly present it should be more common.

DR. BULL said it was always present even in children, but most people did not observe carefully.

UVEAL CYSTS OF THE IRIS.

This case was narrated by Messrs. EALES (Birmingham) and SINCLAIR (Ipswich). In a man, aged 47, the condition in

the left eye was one of absolute glaucoma, the result of a chronic non-inflammatory glaucoma of some years' standing. Extending into the pupillary area from behind the iris were two dark brown globular masses. These moved freely with every movement of the eyeball, and on close examination with a magnifying glass fine jelly-like quivering of the surface of each mass was noted. The diagnosis of cystic detachment of the posterior uveal layer of the iris was fully borne out by pathological examination. This was the first case, so far as the authors knew, in which cystic detachment of the uveal layer of the iris had been diagnosed clinically, though reference was made to other published cases in which this condition was probably present, and attention was drawn to the fine creasing and quivering of the cyst-wall as a diagnostic sign between this condition and pedunculated sarcoma of the iris.

Some remarks were made by MR. TREACHER COLLINS.

OPTIC NERVE ATROPHY IN THREE BROTHERS.

DR. F. M. OGLIVIE read this paper. The brothers were aged 24, 22, and 27; in the first one the sight was said to have failed in one night, in the second in three months, in the third in six months. The had all been smokers, but one had given it up more than seven years before. In all of them there was a central scotoma; in two there was defective color vision; in one only the visual fields for white were contracted. The ophthalmoscopic appearances were not very well marked in two of the brothers; there was pallor of the disc, but the atrophy was not of a very high degree in any of them; the vision was much reduced. One of them had improved somewhat under strychnine and galvanic treatment. A peculiarity in the brothers which they shared with others in the family was the tortuosity of their retinal vessels. So far as the family history could be traced there were no others who had optic nerve atrophy. The father and mother had good sight; the latter was one of ten, and there was no history of defective sight among her brothers and sisters. She had sixteen pregnancies, and fourteen children born alive; all the males except the three who were the subject of this paper had died in infancy.

Remarks were made by Messrs. EDGAR BROWNE and JOHNSON TAYLOR.

Card Specimens.

The following were shown: Mr. Simeon Snell, Alveolar Carcinoma of the Upper Eyelid. Mr. C. D. Marshall, (1) Removal of Metallic Chip from the Vitreous by the Electro-Magnet; Result eighteen months afterwards; (2) Cholesterine in the Anterior Chamber. Mr. W. Adams Frost, Peculiar Ring-Opacity of the Cornea. Messrs. Holthouse and Batten, Peculiar (? congenital) Condition of Optic Disc in a Case of Choroido-Retinitis. Mr. Fischer, Extraction of Lenses in Buphthalmos.—*British Medical Journal.*

A Meeting of Western Oculists, Aurists, Laryngologists and Rhinologists has been called for April 7, in Kansas City, at the Midland Hotel, with a view of organizing a permanent society.

It is with deep regret that we chronicle the death of Dr. James P. Parker, editor of the *Annals of Ophthalmology and Otology*, at his residence in St. Louis, on February 1. After locating in the West he did little in the way of private practice but devoted his whole time and energy to the interests of the *Annals of Ophthalmology and Otology*, which he founded five years ago. His first knowledge of ophthalmology was obtained in the eye clinic of the late Dr. Little, of Philadelphia.

MISCELLANY.

CASE OF DIPHTHERIAL CONJUNCTIVITIS IMPLICATING BOTH CORNEÆ TREATED BY ANTITOXIN.

By W. M. HAMILTON, MD., Patricroft, Lancashire. AND A. EMYRS-JONES, M.D., Surgeon, Manchester Royal Eye Hospital.

On February 28, I found M. M., aged one year, suffering from a mild attack of scarlatina, with temperature 103°, rash well marked, and no throat symptoms. The rash disappeared, temperature fell, and desquamation began on March 4. On March 16, the upper lid of the right eye became swollen, the skin smooth and shining. The margins of the eyelids were red. and there was photophobia. Next day the conjunctiva was very vascular and dotted with red spots. The left eye similarly affected. Ordered a sedative astringent lotion. Next day the lids could be everted only with the greatest difficulty; the conjunctiva was yellow, smooth, and infiltrated with a thick fibrinous exudation. I ordered lotio hydrargyri perchloridi 1 in 5,000 every hour. Next day the corneæ manifested an exudative opacity, and a marginal ulcer appeared on the right cornea, extending to one-sixth of its circumference. The bottom of this ulcer was covered with a yellow opacity. Dr. A. Emyrs-Jones now saw the case with me. He diagnosed diphtherial conjunctivitis. He advised the application of nitrate of silver, gr. xx-℥j every three hours, and that the eyes should be syringed with solution of boro-glyceride. On March 21, there was evidence of the throat being affected, a large diphtheritic patch appearing on the tonsil. That evening, by the advice of Dr. Emrys-Jones, I injected 10 c.cm. diphtheria antitoxin. By next next morning the temperature had fallen from 102° to 99.2°, the swelling of the lids was less and a piece of membrane was detached from the lids. I again injected the antitoxin, and again with marked improvement. The membrane disappeared from the throat and from the lids.

We still continued the application of nitrate of silver and boro-glyceride. On March 26, as there was a recurrence of the membrane in the throat, I again injected the antitoxin. From this time there was no relapse, and the cornea began to clear. The condition of both eyes at this time was inability to open the lids, great vascularity and redness of the conjunctiva, purulent discharge, and complete opacity of the corneæ. We continued the nitrate and boro-glyceride, and instilled atropine every two hours, substituting eserine at night and in the morning. The layers of the cornea separated and came away in the syringing. Unfortunately the infiltration of the right cornea was so deep that perforation took place. The left eye has cleared up entirely with the exception of a very small nebula at the lower margin, which is rapidly disappearing. The sight in this eye is perfect; the child can pick up crumbs from the floor, and can see the normal distance. The right cornea is also clearing, and we are hopeful that in time we may by an iridectomy restore full vision to the eye.

We report this case on account of the extreme rarity of preservation of sight in this fortunately rare and dreadful disease.—*British Medical Journal.*

BOOKS AND PAMPHLETS.

FUNCTIONAL EXAMINATION OF THE EYE. By J. Herbert Claiborne, Jr., M.D. With 21 Illustrations. Philadelphia: The Edwards & Docker Company. 1895. Price, $1.00.

A very clearly written, neat volume, which we can highly recommend to students. Alt.

PAMPHLETS.

"Color Testing." By Ch. H. Williams, M.D.

"The Clinical Chronicle." By E. E. Sattler, M.D.

"Address in Otology." By Lewis H. Taylor, M.D.

"Rhinological Don'ts." By. E. J. Birmingham, M.D.

"Nephritis of the Newly-Born." By A. Jacobi, M.D.

"Manhattan Eye and Ear Hospital Reports," January.

"Hypertrophic Rhinitis." By E. J. Bermingham, M.D.

"Nineteenth Report of the Buffalo Eye and Ear Infirmary."

"P. Blackiston, Son & Co.'s Physician's Visiting List for 1896."

"Affections of the Sound-Perceiving Apparatus." By E. B. Dench, M.D.

"Lumbar Puncture of the Subarachnoid Space." By G. W. Jacoby, M.D.

"Twenth-Seventh Annual Report of the Brooklyn Eye and Ear Hospital."

"Amblyopia from Suppression of Visual Image." By W. B. Johnson, M.D.

"Conservative Treatment of Wounds of the Eyeball." By L. H. Taylor, M.D.

"Seventieth Annual Report of the Massachusetts Charitable Eye and Ear Infirmary."

"A Case of Syringo-Myelia and Its Diagnostic Difficulties." By E. C. Runge, M.D.

"What Shall a General Practitioner do for an Acute Otitis?" By F. B. Dench, M.D.

"Transplantation of Skin in Plastic Operations on the Eyelid." By W. B. Johnson, M.D.

"The Treatment of Acute Inflammation of the Middle Ear and Mastoid." By E. B. Dench, M.D.

"Observations cliniques sur le traitement chirurgical du strabisme" (Clinical observations on the surgical treatment of strabismus). By E. Landolt, M.D.

PLATES TO DR. ALT'S PAPER.

FIG. 1.

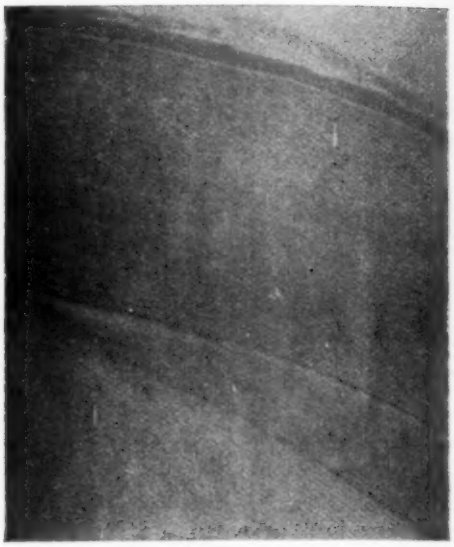

FIG. 2.

FIG. 3.

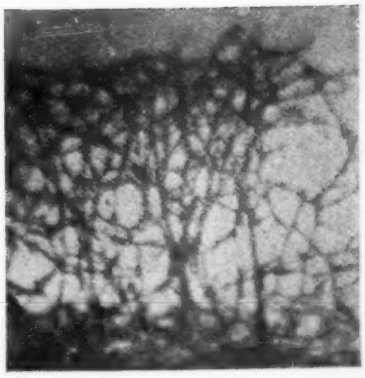

FIG. 4.

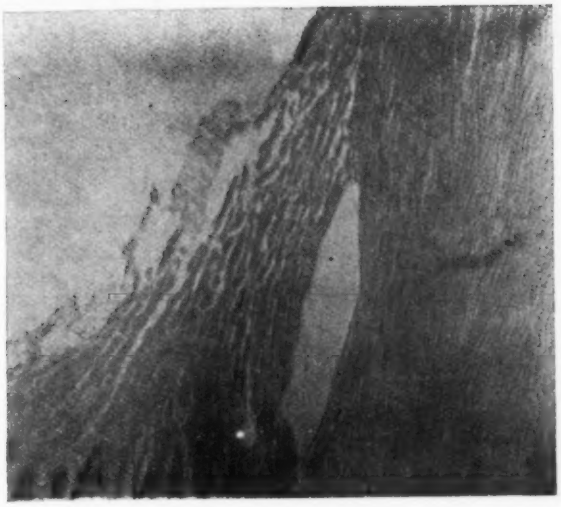

FIG. 5.

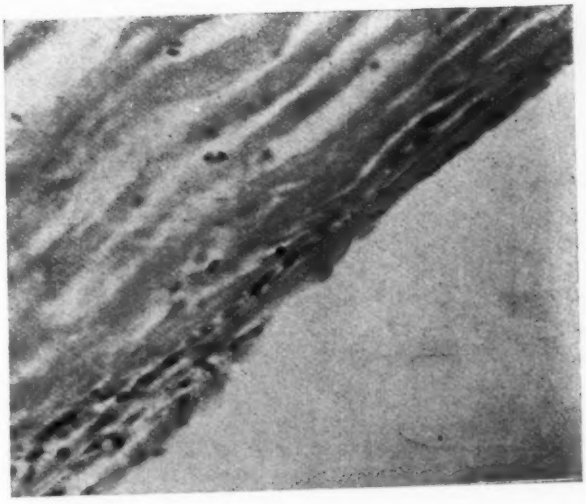

FIG. 6.

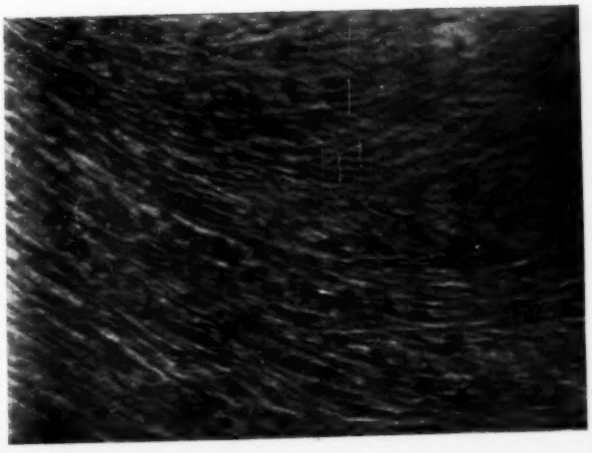

FIG. 7.